What is claimed is:

1. A method of screening an RTA for its capacity to affect lipodystrophy or dyslipidemia in a patient, comprising

(a) administering the RTA to a mesenchymal stem cell or pre-adipocyte cell under culture conditions appropriate for adipogenesis; and

(b) monitoring the cell for an inhibition of adipogenesis;

whereby inhibition of adipogenesis indicates the RTA has the capacity to increase lipodystrophy or dyslipidemia in the patient.

2. The method of claim 1, wherein the RTA is administered to a mesenchymal stem cell.

3. A method of screening an RTA for its capacity to affect lipodystrophy or dyslipidemia in a patient comprising:

(a) administering the RTA to a cell capable of metabolizing lipids under conditions permissible for lipogenesis; and

monitoring net lipogenesis in the cell, whereby a change in net lipogenesis in the cell indicates the protease inhibitor can affect lipodystrophy or dyslipidemia, thereby screening the RTA for its capacity to affect lipodystrophy or dyslipidemia in the patient.

4. A method of screening an RTA for its capacity to affect lipodystrophy or dyslipidemia in a patient comprising:

(a) administering the RTA to a cell capable of metabolizing lipids under conditions permissible for lipolysis; and

(b) monitoring net lipolysis in the cell, whereby a change in net lipolysis in the cell indicates the protease inhibitor can affect lipodystrophy or

dyslipidemia, thereby screening the RTA for its capacity to affect lipodystrophy or dyslipidemia in the patient

5. The method of any of claims 3 or 4, wherein the cell to which the RTA is administered is selected from the group consisting of a mesenchymal stem cell, a liver cell, a muscle cell, an osteoblast, a Schwann cell, an adipocyte, and a pre-adipocyte.

6. The method of any of claims 1, 3 or 4, wherein the RTA is a protease inhibitor.

7. The method of any of claims 1, 3 or 4, wherein the RTA is a NRTI.

8. The method of any of claims 1, 3 or 4, wherein the culture conditions comprise culturing the cell in the presence of a receptor ligand selected from the group consisting of a PPARγ ligand, a RXR ligand, a retinoic acid receptor ligand, insulin, an insulin-like growth factor, a glucocorticoid receptor ligand, and a cAMP-elevating agent.

9. The method of claim 8, wherein the receptor ligand is a PPARγ ligand.

10. The method of claim 9, wherein the PPARγ ligand is an agonist of PPARγ.

11. The method of claim 10, wherein the PPARγ agonist is a thiazolidinedione.

12. The method of claim 8, wherein the receptor ligand is a RXR ligand.

13. The method of claim 12, wherein the RXR ligand is an agonist of RXR.

14. The method of claim 13, wherein the RXR agonist is LGD1069, LG100268, 9-cis retinoic acid, or all-trans retinoic acid.

15. The method of claim 8, wherein the receptor ligand is a retinoic acid receptor ligand.

16. The method of claim 15, wherein the retinoic acid ligand is CH55, 9-cis retinoic acid, or all-trans retinoic acid.

17. The method of claim 8, wherein the receptor ligand is insulin.

18. The method of claim 8, wherein the receptor ligand is an insulin-like growth factor.

19. The method of claim 6, wherein the protease inhibitor is an aspartyl protease inhibitor.

20. The method of claim 19, wherein the aspartyl protease inhibitor is a viral aspartyl protease inhibitor.

21. The method of claim 20, wherein the viral aspartyl protease inhibitor is an HIV protease inhibitor.

22. The method of claim 7, wherein the NRTI is an HIV NRTI.

23. The method of any of claims 2 or 5, wherein the mesenchymal stem cell has the characteristics of a C3H10T1/2 cell.

24. The method of claim 23, wherein the mesenchymal stem cell is a mammalian primary cell.

25. The method of claim 24, wherein the mammalian primary cell is a human primary cell.

26. A method of screening an RTA for its capacity to affect lipodystrophy or dyslipidemia in a patient comprising:

(a) administering the RTA to a cell capable of metabolizing lipids under conditions permissible for metabolizing lipids; and

(b) monitoring the expression of a PPARγ:RXR-regulated gene in the cell, whereby a change in gene expression of the PPARγ:RXR-regulated gene indicates the RTA can affect lipodystrophy or dyslipidemia, thereby screening the RTA for its capacity to affect lipodystrophy or dyslipidemia in the patient.

27. The method of claim 26, wherein the cell capable of metabolizing lipids is selected from the group consisting of a mesenchymal stem cell, a liver cell, a muscle cell, an osteoblast, a Schwann cell, an adipocyte, and a pre-adipocyte.

28. The method of claim 26, wherein the RTA is a protease inhibitor.

29. The method of claim 26, wherein the PPARγ:RXR-regulated gene is a gene which encodes aP2.

30. The method of claim 26, wherein the PPARγ:RXR-regulated gene is a gene which encodes lipoprotein lipase.

31. A method of screening a PI for its capacity to affect lipodystrophy, dyslipidemia, or retinoid-associated toxicity in a patient comprising:

(a) administering the PI to a cell containing a retinoid-regulated gene in the presence of a retinoid; and

(b) monitoring the cell for a change in the expression of the retinoid-activated gene, whereby a change in the expression of the retinoid-

activated gene indicates the PI can affect affect lipodystrophy, dyslipidemia, or retinoid-associated toxicity, thereby screening the PI for its capacity to affect affect lipodystrophy, dyslipidemia, or retinoid-associated toxicity in the patient.

32. The method of claim 31, wherein the cell is an adipocyte or a preadipocyte.

33. The method of claim 31, wherein the PI is an HIV PI.

34. The method of claim 31, wherein the retinoid-activated gene is a gene which encodes alkaline phosphatase.

35. A method of screening a compound for its potential to effect fat metabolism comprising:

(a) contacting a PPARγ receptor-ligand complex with the compound; and

(b) monitoring the complex for displacement of the receptor ligand from the complex, whereby a compound that displaces the receptor has a potential to effect fat metabolism, thereby screening the compound for its potential to effect fat metabolism.

36. A method of screening a compound for its potential to effect fat metabolism comprising:

(a) contacting a PPARγ receptor-ligand complex with the compound; and

(b) monitoring the complex for binding of the compound to the complex, whereby a compound that binds to the complex receptor has a potential to effect fat metabolism, thereby screening the compound for its potential to effect fat metabolism.

37. The method of claim 35 or claim 36, wherein the compound is screened for potential protease inhibitor activity.

38. The method of claim 35 or claim 36, wherein the receptor ligand is a PPARγ ligand.

39. The method of claim 38 wherein the PPARγ ligand is a thiazolidinedione.

40. The method of claim 38, wherein the ligand is BRL49653.

41. A method of screening an RTA for its capacity to affect lipodystrophy or dyslipidemia in a patient, comprising:

(a) administering the RTA to a mammal susceptible to diet-induced obesity; and

(b) monitoring the mammal for an increase in serum lipids, whereby the increase in net serum lipids indicates the RTA has the capacity to increase lipodystrophy or dyslipidemia in a patient.

42. The method of claim 41, wherein the change in serum lipids is indicated by a change in serum triglycerides, free fatty acids, glycerol, or cholesterol.

43. A method of screening an RTA for its capacity to affect lipodystrophy or dyslipidemia in a patient comprising:

a) administering the RTA to a mammal susceptible to diet-induced obesity; and

b) monitoring net fat deposition in the mammal, whereby a change in net fat deposition indicates the RTA can affect lipodystrophy or dyslipidemia, thereby screening the RTA for its capacity to affect lipodystrophy or dyslipidemia in a patient.

44. The method of claim 43, wherein the change in net fat deposition is indicated by

a change in the weight of fat pads.

45. The method of claim 43, wherein the change in net fat deposition is indicated by a change in expression or activity of proteins produced by adipocytes.

46. The method of claim 43, wherein the fat deposition results in interscapular or epididymal fat depots.

47. A method of screening an RTA for its capacity to affect lipodystrophy or dyslipidemia in a patient, comprising:

(a) administering the RTA to a mammal susceptible to diet-induced obesity; and

(b) monitoring the mammal for an increase in blood urea nitrogen or glucose, whereby the increase in blood urea nitrogen or glucose indicates the RTA has the capacity to affect lipodystrophy or dyslipidemia in a patient.

48. A method of screening an RTA for its capacity to affect lipodystrophy, dyslipidemia or retinoid associated toxicities in a patient, comprising:

(a) administering the RTA to a cell containing a retinoid-regulated gene in the presence of a retinoid; and

(a) monitoring the mammal for a change in the expression of a retinoid-activated gene, whereby a change in the expression of the retinoid-activated gene indicates the RTA can affect lipodystrophy, dyslipidemia, or retinoid associated toxicities, thereby screening the RTA for its capacity to affect lipodystrophy, dyslipidemia, or retinoid associated toxicities in a patient.

49. The method of any of claims 41, 43, 47, or 48, wherein the RTA is a protease

inhibitor.

50. The method of any of claims 41, 43, 47, or 48, wherein the mammal is maintained under high-fat diet conditions.

51. The method of any of claims 41, 43, 47, 48, 0r 58 wherein the mammal is a mouse.

52. The method of claim 41, 43, 47, 48, or 58, wherein the mouse has the obesity-related characteristics of a AKR/J mouse.

53. The method of claim 48, wherein the retinoid-activated gene is a gene which encodes alkaline phosphatase.

54. The method of claim 48, wherein the retinoid-activated gene is activated by a retinoid nuclear receptor.

55. A transgenic mouse whose somatic cells comprise and express a transgene conferring sensitivity to an RTA, wherein the total native and transgene expressed in the transgenic mouse is higher than the native gene expressed in a non-transgenic mouse, which transgenic mouse has a phenotype of increased sensitivity to the RTA.

56. A transgenic mouse whose somatic cells comprise and overexpress ubiquitously in all tissues a transgene conferring sensitivity to an RTA, wherein the total native and transgene expressed in the transgenic mouse is higher than the native gene expressed in a non-transgenic mouse, which transgenic mouse has a phenotype of increased sensitivity to the RTA.

57. The transgenic mouse of claim 55 or 56, wherein the RTA is a protease inhibitor.

58. A method of identifying a compound for treating RTA-induced lipodystrophy or dyslipidemia in a mammal, comprising administering the compound to an RTA-sensitive mouse, and monitoring the mouse for a change in the expression of a gene and/or the activity of a gene product associated with lipodystrophy or dyslipidemia, a change in fat distribution, and/or a change in serum lipids, whereby a change in the change in the expression of the gene and/or the activity of the gene product, an increase in fat distribution, or a decrease in serum lipids indicates the compound has the capacity to decrease lipodystrophy or dyslipidemia in the mammal and thereby treat RTA-induced lipodystrophy or dyslipidemia in a mammal.

59. The method of claim 58, wherein the RTA is a HIV protease inhibitor, HIV NRTI or HIV NNRTI.

60. A method of detecting a capacity of a compound to cause RTA-induced lipodystrophy or dyslipidemia in a mammal, comprising administering the compound to an RTA-sensitive mouse, monitoring the mouse for a change in expression of a gene and/or the activity of a gene product associated with lipodystrophy, dyslipidemia or retinoid associated toxicities in the mouse, a change in fat distribution, and/or a change in serum lipids, whereby a change in the expression of the gene and/or the activity of the gene product, an increase in fat distribution, or a decrease in serum lipids indicates the compound has the capacity to cause RTA-induced lipodystrophy, dyslipidemia or retinoid associated toxicities in the mammal.

61. The method of claim 60, wherein the RTA is a protease inhibitor.

62. A method of classifying a patient as being susceptible to RTA-induced lipodystrophy or dyslipidemia, comprising administering RTA to the patient, monitoring the patient for a change in the expression of a gene and/or the activity of a gene associated with lipodystrophy, dyslipidemia or retinoid associated toxicities, a change in fat distribution, and/or a change in serum lipids, whereby a change in the expression of the gene and or the activity of the gene product, an increase in fat distribution, and/or a decrease in serum lipids indicates the patient may be susceptible to lipodystrophy or dyslipidemia; thereby classifying the patient as being susceptible to RTA-induced lipodystrophy, dyslipidemia or retinoid associated toxicities.

63. The method of claim 62, wherein the RTA is a HIV protease inhibitor, HIV NRTI, HIV, NNRTI.

64. The method of any of claims 58, 60, or 62, wherein the RTA is an HIV protease inhibitor.

65. The method of any of claims 58, 60, or 62, wherein the gene is a retinoid-activated gene.

66. The method of any of claims 58, 60, or 62, wherein the gene is activated by a retinoid nuclear receptor.

67. The method of any of claims 58, 60, or 62, wherein the gene is a PPARγ:RXR-activated gene.

68. The method of any of claims 58, 60, or 62, wherein the gene is a protease inhibitor regulated gene.

69. The method of any of claims 58, 60, or 62, wherein the change in gene expression comprises an increase in gene expression.

70. The method of any of claims 58, 60, or 62, wherein the change in gene expression comprises a decrease in gene expression.

Add A2.